1-1-2007

Comparison of the effects of continuous and pulsed mobile phone like RF exposure on the human EEG

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Keywords

exposure, effects, continuous, pulsed, human, phone, like, rf, comparison, mobile, eeg

Disciplines

Arts and Humanities | Life Sciences | Medicine and Health Sciences | Social and Behavioral Sciences

Publication Details

Perentos, N., Croft, R. J., McKenzie, R. J., Cvetkovic, D. & Cosic, I. (2007). Comparison of the effects of continuous and pulsed mobile phone like RF exposure on the human EEG. Australasian Physical and Engineering Sciences in Medicine, 30 (4), 274-280.

SCIENTIFIC NOTE

Comparison of the effects of continuous and pulsed mobile phone like RF exposure on the human EEG

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Abstract

It is not clear yet whether Global System for Mobiles (GSM) mobile phone radiation has the ability to interfere with normal resting brain function. There have been reports that GSM exposure increases alpha band power, and does so only when the signal is modulated at low frequencies (Huber, R., Treyer, V., Borbely, A. A., Schuderer, J., Gottselig, J. M., Landolt, H.P., Werth, E., Berthold,T., Kuster, N., Buck, A and Achermann, P. Electromagnetic fields, such as those from mobile phones, alter regional cerebral blood flow and sleep and waking EEG. J Sleep Res 11, 289-295, 2002.) However, as that research employed exposure distributions that are not typical of normal GSM handset usage (deep brain areas were overexposed), it remains to be determined whether a similar result patterning would arise from a more representative exposure. In this fully counterbalanced cross-over design, we recruited 12 participants and tried to replicate the modulation linked post exposure alpha band power increase described above, but with an exposure source (dipole antenna) more closely resembling that of a real GSM handset. Exposures lasted for 15 minutes. No changes to alpha power were found for either modulated or unmodulated radiofrequency fields, and thus we failed to replicate the above results. Possible reasons for this failure to replicate are discussed, with the main reason argued to be the lower and more representative exposure distribution employed in the present study. In addition we investigated the possible GSM exposure related effects on the non-linear features of the resting electroencephalogram using the Approximate Entropy (ApEn) method of analysis. Again, no effect was demonstrated for either modulated or unmodulated radiofrequency exposures.

Key words EEG, GSM, ApEn, alpha band

Introduction

The GSM telecommunications system has been in use by the public for more than a decade. It is not clear yet whether any biological effects arise from the exposure to the radiofrequency electromagnetic fields (RF) emitted by GSM handheld devices. Public concern about possible effects of mobile phone exposures on health is still present, which highlights the need to address the uncertainty. There are currently no accepted physical mechanisms with which to explain possible bioeffects at the low energy levels that mobile phones emit, and so empirical tests of the possibility are required to address this issue.

Numerous studies have been published looking at different aspects of brain function under GSM exposure. These indices include event related potentials and performance tasks[1], sleep variables[2] and resting electroencephalogram (EEG) variables[3]. On reviewing these studies[4,5] it can be observed that results are highly variable. Studies that attempt to replicate results often fail[1,6], with at least one exception[7,8]. Part of the variance between studies arises due to the variable experimental conditions employed. These include exposure duration, post exposure monitoring periods and statistical analysis methods. Therefore, the need to thoroughly investigate these effects remains.

One of the most consistent findings reported across several independent studies is that of increased resting EEG alpha band activity[3,7,8]. The consistency observed across these studies could be partly attributed to some common methodologies employed such as accurate dosimetry, the extended duration of exposure and the duration of EEG recording. The significance of the last two factors is discussed comprehensively elsewhere[9]. On the contrary, studies that investigated resting EEG but did not show an effect on alpha band activity (negative finding) have tended to employ small exposure durations, for example Roschke and Mann[10], who used exposure periods shorter than 3.5 minutes.

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Received: 20 July 2007; Accepted: 3 October 2007

Another important experimental condition which varies between studies is that of the characteristics of the electromagnetic stressor. Of particular importance are the Specific Absorption Rate (SAR) distributions in the head and the spectral content of the stressor due to the type of modulation employed. At frequencies greater than 100 kHz, SAR is defined as the rate at which electromagnetic energy is absorbed per unit mass of biological tissue. It is the main metric for assessing exposure compliance of devices such as GSM mobile phone handsets.

With respect to SAR distributions, Curcio *et al.*[8] and Croft *et al.*[7] utilised real GSM handsets with monopole antennas as opposed to Huber *et al.*[3] who used a non-handset based patch antenna. In Huber *et al.* 2003[11] it was shown that using a patch antenna, such as the one used in Huber *et al.*[3], results in a SAR variation within the exposed hemisphere of approximately 12.5 dB (between the exposed surface and the farthest side to the exposed hemisphere), with some areas deep in the brain exposed to SARs of at least -5dB. However, it has been shown that the variation of SAR in the head due to exposure from monopole antennas operating at 900MHz is much greater, reaching 24 dB[12]. Thus it is evident that a more homogeneous exposure is produced under the patch antenna relative to the localised nature of the exposure resulting from a monopole antenna. Huber *et al.*[3] argue that the SAR observed from the patch antenna they used would be representative of a real mobile phone exposure. However, as discussed above, this is not the case, and in particular, some parts of the mid-brain are exposed to much higher SARs (by a factor of up to 80) than would occur from any commercially available mobile phone.

Absence of any modulations results in Continuous Wave (CW) exposures, while the presence of GSM-like modulation results in Pulse Modulated (PM) exposures. It has been suggested[13] that the presence of modulations, and in particular, low frequency spectral content, might be important in order to induce biological effects. Consistent with this, recent positive studies have all employed PM exposure schemes. We should note that the nature of pulse modulation and spectral content amongst some of these studies did differ. For example Huber *et al.*[3] utilised a signal with the same frequency components as the Discontinuous Transmission (DTX)[14] spectrum of the GSM handset (2, 8, 217 and 1736Hz) while Curcio *et al.*[8] and Croft *et al.*[7] made use of the main 217 and 1736 Hz pulse modulation frequencies. In addition, in Croft *et al.* the pure Extremely Low Frequency (ELF) exposures (217 and 1736Hz) were also present due to real handset battery operation.

Huber *et al.*[3] tested the importance of modulation characteristics by comparing effects on the EEG arising after exposure to PM RF (DTX like spectrum) with those arising after CW RF exposure. Effects were observed on alpha band activity after PM RF exposure but not after CW RF. They concluded that modulation characteristics are critical in inducing the alpha band activity increase after mobile phone exposure. However, as discussed above, since the exposure distribution used differed from that of a real mobile phone, the question remains of whether such an effect would be observed under an exposure that more closely resembles that of a mobile phone, or whether Huber *et al*'s findings are specific to their exposure setup.

Hence, the main purpose of the present study was to attempt to replicate the findings of Huber *et al.* (CW RF vs PM RF) using a monopole antenna as the exposure source, thereby allowing us to test whether the spectral content is indeed significant under more realistic exposures, and making the findings of Huber *et al.*, if replicated, more generalisable to real mobile phone handsets and thus everyday exposures.

In this paper, we also explored the usefulness of a non-linear analysis method of the EEG. The most conventional way of analysing resting EEG signals is the Fourier transform, which is a linear method of signal analysis in the frequency domain. On the other hand, evidence exist that EEG can behave as a non-linear oscillator[15] and therefore a non-linear signal analysis method may be appropriate. One such method is the Approximate Entropy (ApEn) with which the non-linear measure of complexity of the EEG can be calculated. This may provide new information regarding the interaction of RF with the EEG that would otherwise remain undetected with conventional linear analyses methods.

For example, the regularly occurring pulsing of the incident radiation could introduce more regularity in the EEG time series and therefore decrease the complexity of the resting EEG, much like the case of auditory or visual entrainment[16,17]. In at least one case[18] an effect on the entropy of the EEG of rats has been shown under the influence of low frequency pulses; although in that case the electromagnetic fields were of much higher intensity and of different frequencies to those investigated here.

Materials and methods

Subjects

Twelve healthy volunteers (6 males and 6 females) aged between 19 and 32 (Mean = 26.5, SD = 3.29) were recruited. Participants were informed about the details of the experiment and written informed consent was obtained. The design of the study was approved by the RMIT University Human Research Ethics Committee.

Protocol

Participants attended a 2 hour recording session in which RF exposure occurred for two 15-minute intervals (15 minutes for CW and 15 for PM RF), a sufficient time to allow for a cumulative effect to take place[9], as well as there being a 15-minute sham exposure, (Sham). Resting EEG was monitored throughout the experiment while subjects were seated comfortably with eyes closed.

The exposure protocol, depicted in Figure 1, comprised twelve 7 ½ minute periods, four periods for each exposure condition (one pre exposure, two during exposure, one post exposure period). Subjects were instructed to remain as still as possible and keep their eyes closed throughout the

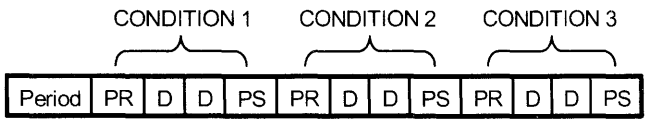

Figure 1. *Experimental protocol. PR: pre-exposure, D: during exposure, PS: post exposure. Conditions 1, 2 and 3 are Sham, PM RF and CW RF randomly assigned. Each period lasts for 7½ minutes. For all subjects, after each PS period, there was a 1 minute break.*

duration of recordings. One-minute breaks were allocated between each 7½ minute period in which subjects were instructed to open their eyes and stretch; the latter aimed at reducing subject fatigue and irritation from being confined for long periods. Although a fully counterbalanced double blind cross over design was applied, the study was analysed as single blind. This was necessary since ELF artefacts were present 'during' exposure, most likely originating from the PM RF signal demodulation in the EEG amplifiers, which were sometimes visible in the EEG recordings. For the same reason, only the pre and post exposure periods are analysed here.

Data acquisition

Participants were fitted with a Compumedics Neuroscan 19 Channel Tin Quick EEG Cap employing the standard 10/20 international electrode positioning system (excluding F_z, C_z and P_z), referenced to linked mastoid electrodes. Data were recorded using the MINDSET, MS-1000, 16-channel EEG amplifier (fixed gain of 32768). Signals were sampled at a rate of 256 samples per second and band pass filtered with two, fourth-order Sallen-Key active filters, 48 dB roll-off per octave and a 3dB pass band between 1.5 Hz - 34 Hz. Impedances were below 12 kΩ at the start of the recording. Recordings took place inside a electromagnetically shielded room, with the RF generator and amplifier situated outside the shielded room. Apart from the subject under test, another person, responsible for monitoring the physiological recording equipment, was present in the shielded room. Physiological recording equipment and modulation circuitry were also inside the room.

RF exposure

Dummy handset

The exposure device used was a model handset consisting of a metallic casing acting as the ground and a monopole antenna; a crude approximation of a commercial GSM handset. Detailed description of the handset can be obtained elsewhere[19, 20]. The main advantage of using such a model handset instead of a real one is that it does not produce thermal and auditory queues during operation, as is the case with real GSM handsets, which could compromise the blinding of the experiments. The handset device was placed according to the standard 'touch' ear to mouth position[21]. A left hemisphere exposure was used for all subjects.

Exposure characteristics

Two different signals were used. The first was an unmodulated RF signal at 900MHz containing no low frequency content (CW RF), and the second was a pulse modulated 900MHz signal containing all components found in the signal emitted by GSM handsets which are operating in the DTX mode (PM RF). These components arise from the frame structure of the GSM signal and include 2, 8, 217 and 1736 Hz plus harmonics[14]. Input levels to the model handset were set to simulate the 2W peak signal of a commercial GSM mobile phone handset operating at 900 MHz. For compliance purposes, dosimetric evaluation of this handset was performed at a commercial facility in Melbourne Australia (EMC technologies). With a peak input power of 276mW, a 10g averaged Peak SAR of 1.56W/kg is achieved at the base of the handset's antenna which corresponds to the left ear region of the SAM phantom.

Data analysis

For the spectral analysis, data was epoched into 2 second segments and analysed in the Matlab environment, using EEGLAB[22]. Epochs were baseline corrected over the entire epoch. Segments containing data greater than +/- 60μV were automatically rejected. On average 67% of the data were retained. To obtain relevant EEG bands, processed epoched time series were passed through a Fourier transform (Hamming window) with a window size of 2 seconds with no overlap. Subsequently, the following EEG bands were extracted: δ (2 - 4 Hz), θ (4.5 - 7 Hz), α (7.5 – 13 Hz) and β (13.5 - 32 Hz). For each band, the maximum spectral power (POWER) is calculated. For the ApEn analysis, 8 artefact free (rejection criteria as above) consecutive seconds for each 7 minute period were retained and subsequently analysed in the Matlab environment.

Statistical analysis

Two Statistical Analyses were performed with SPSS statistical package version 11.5:

Fourier analysis

The first analysis involved measures of Fourier Power Spectra of recorded EEG time series. It was based on the comparison of recordings collected before and after the real exposures, a difference value, with that collected before and after the sham exposure (again expressed as a difference value). For each channel the difference value is obtained by subtracting the 'after' radiation power spectra from the 'before' radiation power spectra. A positive result would indicate an increase in the relevant amplitude level and a negative would indicate a decrease. Electrodes were grouped (by averaging) in pairs in order to reduce the amount of statistical comparisons. Electrode groups were: Left Prefrontal (LPF = mean (Fp1, F7)), Right Prefrontal (RPF = mean (Fp2, F8), Left Frontal (LF = mean (F3, T3)), Right Frontal (RF = mean (F4, T4)), Left Central (LC = mean (C3 & T5)), Right Central (RC = mean (C4, T6), Left Posterior (LP = mean (P3, O1)) and Right Posterior

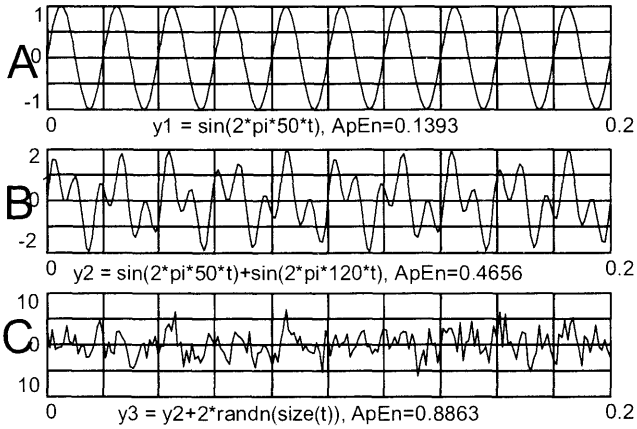

Figure 2. *Three different functions of increasing complexity are compared with respect to their ApEn values (A: simple sinusoidal, B: two different frequency sinusoidals algebraically added and C: function B but embedded in noise). When passed through the ApEn algorithm we obtain the expected relative increase in ApEn from A to C.*

(mean = (P4, O2)). Statistical significance was tested with Repeated Measures Analyses of Variance (RM-ANOVA) for each of the four predefined EEG bands (δ, θ, α, β). Each RM-ANOVA consisted of three within subjects factors; Exposure Source (SRC: Sham, PM RF and CW RF), Laterality (LAT: Left and Right hemisphere) and Sagittality (SAG: Prefrontal, Frontal, Central, Posterior). Where significance was observed, appropriate post hoc tests were conducted with Bonferroni corrections.

ApEn analysis and theory

The second method of analysis was based on ApEn measures, a statistical measure of complexity that can be applied to any kind of finite time series with a minimum data length of 50 samples. It has been used on the analysis of human EEG data in relation to Alzheimer's disease[23], epilepsy[24], hypobaric hypoxia[25] and sleep[26].

Theoretical background

ApEn represents the mean probability that temporal sections (of length *m*) of a time series which are 'close' to each other (where 'close' is any distance smaller than a threshold distance *r*), will remain 'close' for the temporal sections of incremental length *m+1*.

Mathematically, given a time series containing N elements: $\{x(n)\} = x(1), x(2)...x(N)$ we create vector sequences defined as $X(1)...X(N-m+1)$ where $X(i) = x(i), x(i+1)...x(i+m-1)$, $i = 1 \sim N-m+1$ and m is the vector length, in data points, over which comparisons are made.

We define distance d as follows:

$$d[X(i), X(j)] = \max \left| \begin{matrix} x(i)-x(j), x(i+1)-x(j+1), \\ ...x(i+m-1)-x(j+m-1) \end{matrix} \right| \quad \text{where}$$

$j = 1 \sim N-m+1$. Now for a given $X(i)$ we count the number of $X(j)$ s for which $d[X(i), X(j)] \le r$. We denote this as $N^m(i)$. Then we calculate $C_r^m(i) = N^m(i)/N-m+1$ which represents the portion of vectors that obey the similarity criterion, $d \le r$, in comparison to $X(i)$. We repeat this for all $X(i)$ s, obtain $C_r^m = \sum_i^N C_r^m(i) \Big/ N-m+1$ and define $\phi^m(r) = \ln C_r^m$. We repeat the above process for m+1 and finally obtain $ApEn(m,r) = \phi^m(r) - \phi^{m+1}(r)$.

ApEn data analysis

The ApEn measures were calculated using Kaplan *et al.*'s implementation in the Matlab environment[27]. Although it was developed and used for Heart Rate Variability, its use extends for any kind of information series.

Data was first passed through artefact rejection based on the same criteria as the ones used for the Fourier analysis. A data length of 2048 consecutive points, free of artefacts, equivalent to 8 seconds, was retained for each 7 ½ minute period and was then submitted for ApEn analysis. Consistent with Pincus[28] we choose an embedding dimension, *m*, equal to 2 and a filter factor, *r*, equal to 0.2 times the standard deviation of each 8 second interval. To verify the correct performance of the algorithm i.e. increasing ApEn value with corresponding increase in mathematical function complexity we submitted functions of increasing complexity through it. Increasing ApEn values were obtained which demonstrates the efficacy of the algorithm, Figure 2 A One-Way Repeated Measures Analysis of Variance (RM-ANOVA) was performed on the difference values ('after' minus 'before', 2 channel averages for each brain region as defined in the FFT analysis) of ApEn measures obtained using the same within factors that were used for the Fourier Spectral analysis (SRC, LAT, SAG).

Non linearity test

To demonstrate that recorded EEG time series contain non-linear features we perform a non-linearity test which is based on surrogate analysis. The original time series is compared with the surrogate versions of the same data whose assumed non-linear features have been removed. It is noted that a complexity analysis can be performed on the data without evidence of non-linearity. Ten surrogate data series were created for each 8-second recording and the respective ApEn was calculated. Surrogates were created using the Amplitude Adjusted Fourier Transform Method (AAFT). In this, the Fourier amplitude spectrum of the data was calculated and retained, phase was randomised and inverse Fourier was executed. This way surrogates were restricted in terms of linear properties of the original data but were otherwise random. A t-test was performed on each real data series against its ten surrogates. With 9 degrees of freedom and a significance level of 0.05, a critical value of t=2.26 was obtained.

Table 1. *A Summary of the RM-ANOVA results. The factor SRC for the delta band is the only one that reached significance but post hoc contrasts did not reveal any statistically significant diffrences at the Bonferroni-corrected alpha level.*

Band	Factor or factor interaction	Significance (p)
Delta	SRC	*0.038**
	SAG*SRC	0.363
	LAT*SRC	0.234
	LAT*SAG*SRC	0.608
Theta	SRC	0.557
	SAG*SRC	0.809
	LAT*SRC	0.213
	LAT*SAG*SRC	0.746
Alpha	SRC	0.435
	SAG*SRC	0.149
	LAT*SRC	0.103
	LAT*SAG*SRC	0.366
Beta	SRC	0.700
	SAG*SRC	0.549
	LAT*SRC	0.917
	LAT*SAG*SRC	0.447

Table 2. *'p' values for the various factors and factor interactions of the ApEn analysis. None reached or approached significance.*

Factor or factor interaction	Significance.(*p*)
SRC	0.581
SAG * SRC	0.952
LAT * SRC	0.412
SAG * LAT * SRC	0.461

Results

Fourier power spectra

For the main factor SRC, significance was observed in the delta band (p = 0.034) only. Post hoc tests for the factor SRC in the delta band showed that neither Sham compared to PM RF (p=0.502), nor Sham compared to CW RF (p=0.087), reached the Bonferroni-corrected significance level (alpha = 0.002). For each EEG band, no significant effects or interactions were obtained for SRC with SAG, LAT, or SAG by LAT (Table 1). Mean power spectra are shown in Figure 3 and topographies of grand mean spectral powers for before and after exposure periods are shown in Figure 4.

ApEn analysis

Non-linearity tests based on the method of surrogate analysis showed that in total, 80% of cases contained statistically significant non-linear features. One subject specifically showed no evidence of non-linearity in all of its recorded intervals, accounting for the bulk of the data that failed the surrogate test. No significant difference was obtained for any of the factors or their interactions (Table 2). Mean ApEn values are shown in Figure 5.

Discussion

This study was performed with two aims. The first was to compare the possible effects arising from CW RF versus PM RF in an attempt to reproduce the results reported by Huber *et al*[3], but using a radiation exposure distribution more closely resembling that of a GSM mobile phone handset. The secondary aim was to investigate the usefulness of a non-linear analysis method, the ApEn.

We failed to replicate the reported alpha band activity increases during resting wakefulness under PM RF monopole exposure with the conventional Fourier analysis method. The only significant factor was SRC (p = 0.034) for the delta band, but post-hoc tests did not reveal any statistically significant changes at the Bonferroni-corrected significance level. Delta band changes have been previously reported on at least one occasion[9]. The ApEn

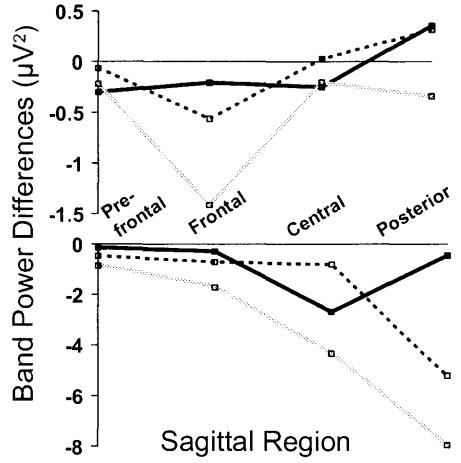

Figure 3. *Band Power differences in μV^2 ('after' minus 'before') on the y-axis for Sagittal values on the x-axis for Sham (solid black line), PM RF (dashed black line) and CW (solid grey line) conditions. Top graph for delta band and bottom for alpha band, both for left hemisphere (ipsilateral to exposure).*

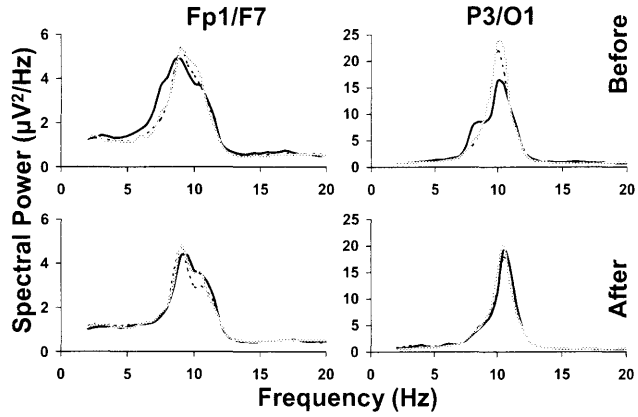

Figure 4. *Grand mean EEG spectral power before (top) and after exposure (bottom) are shown at frontal and occipital regions, Sham (solid black line), PM RF (dashed black line) and CW (solid grey line) exposures.*

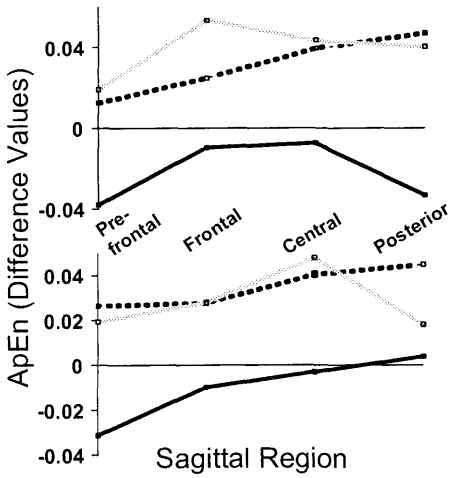

Figure 5. *Means of ApEn difference values on the y-axis with Sagittal regions on the x-axis. Sham (solid black line), PM RF (dashed black line) and CW (solid grey line), Top panel: Ipsi-lateral to exposure and Bottom panel: contra-lateral to exposure.*

method did not reveal any statistically significant differences in the EEG with relation to the exposure condition.

The use of the ApEn analysis method provided the opportunity to investigate the non-linear features of the recorded EEG data and any correlation with the RF exposure. It is difficult to determine whether this represents a lack of advantage of the ApEn measure over FFT, or whether differences between the methods may have been masked by the lack of any significant effect. For instance, it is possible that the effect sizes for both methods were too small to be detected with the current sample size, with larger sample sizes required to clarify this issue.

Three factors are identified which may have contributed to the inconsistency between findings in the present study and those reported in Huber *et al.*[3]

Firstly, the difference in SAR distributions between the two antennas could have been a contributing factor. As discussed in the introduction, the patch antenna source used by Huber *et al.*[3] covers exposures for all possible mobile phone locations relative to the brain simultaneously (equivalent to superimposing exposures from more than one handset). The homogeneity of exposure results in over-exposure of areas deep in the brain, which could possibly be more vulnerable to external stressors such as RF and thus result in an enhanced effect. In support of the above, the effect in Huber *et al.*[3] was shown with a smaller (N = 15) sample size than the effect reported by Curcio *et al.*[8] and Croft *et al.*[7] (sample sizes of 20 and 120 respectively), with the latter researchers employing a realistic exposure which would produce a lower exposure in deep brain regions. In addition Curcio *et al.* and Croft *et al.* report effects during radiation but not after, as opposed to Huber *et al.* who report a post exposure effect larger than the during exposure effect of Croft *et al.* and Curcio *et al.* It may be speculated that the patch antenna exposure of Huber *et al.* produced an effect that lasted longer than the effect from the monopole exposure.

Secondly, the exposure duration herein was 15 minutes as opposed to Huber *et al.* who used 30 minutes. More time would allow for a cumulative process, if one exists, to enhance the effect under the longer exposure, thus justifying the observed effect under the Huber *et al.* protocol as opposed to the protocol used here. On the other hand effects have been previously demonstrated with exposure lengths of 15 minutes[29] and smaller[30] a fact that makes the above argument a less likely explanation for the observed discrepancy. A third factor is that of possible carry-over effects since exposures took place in the same day. This may have increased error variance and reduced the chance of identifying a real effect.

Finally, when comparing the results of Curcio *et al.*[8] and Croft[7,9] with those of Huber *et al.*[3] we observe that the former two made use of the 217 Hz modulation and 1736Hz only, whereas Huber *et al.*[3] made use of a DTX like spectrum (2, 8, 217, 1736 Hz). Thus if the 2 and 8Hz components contributed to the increase in alpha, this would be another factor that could account for the larger effect size observed by Huber *et al.*[3]

Based on the assumption that the effect expected from a monopole antenna would be of a smaller effect size in comparison to that from a large patch antenna, and given that the small sample size of the present study has limited interpretation, we will now conduct a study with a substantially larger sample size in order to compare CW and PM RFs, with exposure distributions resembling those from real mobile phones.

In conclusion, this study failed to replicate the findings of Huber *et al.*[3] Possible reasons for this have been identified, with the main reason argued to be the difference between the homogenous exposure of Huber *et al.* and the monopole exposure of the present study. That is, although in homogeneous exposures (e.g. patch antenna source) the modulation content has been shown to be important in affecting the resting EEG, the same may not be true for more realistic exposures such as the dipole source used here. In addition, the exploration of a non-linear feature of complexity of the EEG did not produce greater clarification of this issue.

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